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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Apr 08	"Ask CAS" for self-help around the clock
NEWS 3	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 4	Apr 09	ZDB will be removed from STN
NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS 14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS 15	Jul 30	NETFIRST to be removed from STN
NEWS 16	Aug 08	CANCERLIT reload
NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
NEWS 19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS 20	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 23	Sep 03	JAPIO has been reloaded and enhanced
NEWS 24	Sep 16	Experimental properties added to the REGISTRY file
NEWS 25	Sep 16	CA Section Thesaurus available in CAPLUS and CA
NEWS 26	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS 27	Oct 21	EVENTLINE has been reloaded
NEWS 28	Oct 24	BEILSTEIN adds new search fields
NEWS 29	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 30	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS 31	Nov 18	DKILIT has been renamed APOLLIT
NEWS 32	Nov 25	More calculated properties added to REGISTRY
NEWS 33	Dec 02	TIBKAT will be removed from STN
NEWS 34	Dec 04	CSA files on STN
NEWS 35	Dec 17	PCTFULL now covers WP/PCT Applications from 1978 to date
NEWS 36	Dec 17	TOXCENTER enhanced with additional content
NEWS 37	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 38	Dec 30	ISMEC no longer available
NEWS 39	Jan 13	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 40	Jan 21	NUTRACEUT offering one free connect hour in February 2003
NEWS 41	Jan 21	PHARMAML offering one free connect hour in February 2003
NEWS 42	Jan 29	Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC

NEWS EXPRESS	January 6 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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DICTIONARY FILE UPDATES:   2 FEB 2003  HIGHEST RN 484639-64-7

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

  Please note that search-term pricing does apply when
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Experimental and calculated property data are now available.  See HELP
PROPERTIES for more information.  See STNote 27, Searching Properties
in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> e perillyl alcohol/cn
E1            1      PERILLUP KETOL/CN
E2            1      PERILLYL ACETATE/CN
E3            1  --> PERILLYL ALCOHOL/CN
E4            1      PERILLYL ALCOHOL DEHYDROGENASE/CN
E5            1      PERILLYL ALDEHYDE/CN
E6            1      PERILLYL BROMIDE/CN
E7            1      PERILLYL CHLORIDE/CN
E8            1      PERILLYLACETALDEHYDE/CN
E9            1      PERIMARGINE/CN
E10           1      PERIMARGINE, HEXAHYDRO-/CN
E11           1      PERIMED/CN
E12           1      PERIMETAZINE/CN
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=> s e3
L1              1 "PERILLYL ALCOHOL"/CN

=> d
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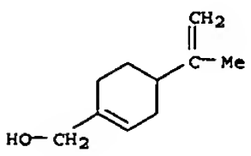
L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 536-59-4 REGISTRY
CN 1-Cyclohexene-1-methanol, 4-(1-methylethenyl)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN p-Mentha-1,8-dien-7-ol (7CI, 8CI)
OTHER NAMES:
CN dl-Perillyl alcohol
CN Perilla alcohol
CN Perillic alcohol
CN Perillol
CN **Perillyl alcohol**
FS 3D CONCORD
DR 7644-38-4, 1406-56-0, 66141-69-3
MF C10 H16 O
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, NAPRALERT, PHAR, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

392 REFERENCES IN FILE CA (1962 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
392 REFERENCES IN FILE CAPLUS (1962 TO DATE)
10 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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=> fil .search
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                6.70       6.91

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=> s l1
L2          679 L1

=> s l2 and (tumor? or tumour? or cancer? or sarcoma?)
L3          247 L2 AND (TUMOR? OR TUMOUR? OR CANCER? OR SARCOMA?)

=> s l3 and sensit?
L4           23 L3 AND SENSIT?

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           17 DUP REM L4 (6 DUPLICATES REMOVED)

=> d ibib ab 1-
YOU HAVE REQUESTED DATA FROM 17 ANSWERS - CONTINUE? Y/(N):y
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L5 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 2002:736903 CAPLUS
DOCUMENT NUMBER: 137:244075
TITLE: Monoterpenes and sesquiterpenes as chemotherapeutic and radiation **sensitizers** and immunomodulators
INVENTOR(S): Gould, Michael N.; Howard, Steven P.; Rajesh, Deepika
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 41 pp., Cont.-in-part of U.S. Ser. No. 878,797.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002137799	A1	20020926	US 2001-14724	20011107
US 2002054850	A1	20020509	US 2001-878797	20010611
PRIORITY APPLN. INFO.:			US 2000-246887P P	20001108
			US 2001-878797 A2	20010611
			US 2000-211506P P	20000614

AB A method of **sensitizing tumor** cells to radiation therapy, chemotherapy and immunomodulatory therapy, comprising the step of exposing the **tumor** cell to an effective amt. of at least one monoterpene or sesquiterpene and treating the **tumor** cell is disclosed.

L5 ANSWER 2 OF 17 USPATFULL
ACCESSION NUMBER: 2002:243049 USPATFULL
TITLE: Measurement of protective genes in allograft rejection
INVENTOR(S): Avihingsanon, Yingyos, Boston, MA, UNITED STATES
Ma, Nalli, Wirchester, MA, UNITED STATES
Strom, Terry B., Brookline, MA, UNITED STATES
Soares, Miguel C., Boston, MA, UNITED STATES
Ferran, Christiane, Brookline, MA, UNITED STATES
Suthanthiran, Manikkam, Scarsdale, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002132235	A1	20020919
APPLICATION INFO.:	US 2001-777732	A1	20010206 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-199327P	20000424 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY, HOAG & ELIOT, LLP, PATENT GROUP, ONE POST OFFICE SQUARE, BOSTON, MA, 02109
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 2820
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to methods of evaluating transplant rejection in a host comprising determining a heightened magnitude of gene expression of genes in rejection-associated gene clusters. The disclosed gene clusters include genes that are substantially co-expressed with cytotoxic lymphocyte pro-apoptotic genes, cytoprotective genes and several other cytokine and immune cell genes.

L5 ANSWER 3 OF 17 USPATFULL
ACCESSION NUMBER: 2002:105649 USPATFULL
TITLE: Monoterpenes and sesquiterpenes as chemotherapeutic **sensitizers** and radiation **sensitizers**
INVENTOR(S): Gould, Michael N., Madison, WI, UNITED STATES
Howard, Steven P., Madison, WI, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002054850	A1	20020509
APPLICATION INFO.:	US 2001-878797	A1	20010611 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-211506P	20000614 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: QUARLES & BRADY LLP, 411 E. WISCONSIN AVENUE, SUITE 2040, MILWAUKEE, WI, 53202-4497
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 475
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of **sensitizing tumor** cells to radiation, comprising the step of exposing the **tumor** cell to an effective amount of at least one monoterpene or sesquiterpene and irradiating the **tumor** cell, is disclosed.

L5 ANSWER 4 OF 17 USPATFULL
ACCESSION NUMBER: 2002:17248 USPATFULL
TITLE: Treatment of hyperproliferative, inflammatory and related mucocutaneous disorders using inhibitors of mevalonate synthesis and metabolism
INVENTOR(S): Parks, Thomas P., San Mateo, CA, UNITED STATES
Grayson, Stephen, San Rafael, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002010128	A1	20020124
APPLICATION INFO.:	US 2001-833384	A1	20010411 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-197357P	20000413 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 47
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1443
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods for treating a variety of hyperproliferative and inflammatory mucocutaneous disorders, including, basal cell carcinoma, squamous cell carcinoma, psoriasis and atopic dermatitis, as well as skin irritation and disorders associated with skin aging and skin photodamage using inhibitors of cholesterol metabolism. The present invention further relates to the discovery that the combined use of several inhibitors of cholesterol metabolism produces synergistic effects. Furthermore, the present invention is directed to the use of inhibitors of cholesterol metabolism as excipients to enhance the effects of antiinflammatory drugs.

L5 ANSWER 5 OF 17 USPATFULL
ACCESSION NUMBER: 2002:217302 USPATFULL
TITLE: Method of suppressing tumor growth with combinations of isoprenoids and statins
INVENTOR(S): Elson, Charles E., Madison, WI, United States
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6441029	B1	20020827
APPLICATION INFO.:	US 2000-587737		20000605 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1998-27546, filed on 23 Feb 1998, now patented, Pat. No. US 6133312

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-39790P	19970304 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Goldberg, Jerome D.
LEGAL REPRESENTATIVE: Quarles & Brady LLP
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 1066
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of inhibiting the growth of tumor cells is disclosed. In one embodiment, this method comprises the step of exposing tumor cells to an effective amount of a composition comprising at least two compounds selected from the group consisting of tocotrienols, statins and ionones.

L5 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:923635 CAPLUS
DOCUMENT NUMBER: 136:34013
TITLE: Monoterpenes and sesquiterpenes as chemotherapeutic **sensitizers** and radiation **sensitizers**
INVENTOR(S): Gould, Michael N.; Howard, Steven P.
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, USA
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001095936	A2	20011220	WO 2001-US18824	20010612
WO 2001095936	A3	20020718		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-211506P P 20000614
AB A method of **sensitizing tumor** cells to radiation, comprising the step of exposing the **tumor** cell to an effective amt. of at least one monoterpene or sesquiterpene and irradiating the **tumor** cell, is disclosed. Examples are given on inhibition of various **tumor** cells (glioma, glioblastoma, prostate **tumor**) by radiotherapy and radiosensitization with perillyl alc., limonene, L-carvone, menthol, citral, myrcene, and geranyl tiglate.

L5 ANSWER 7 OF 17 USPATFULL
ACCESSION NUMBER: 2001:229235 USPATFULL
TITLE: METHOD FOR USING SOLUBLE CURCUMIN TO INHIBIT PHOSPHORYLASE KINASE IN INFLAMMATORY DISEASES
INVENTOR(S): HENG, MADALENE C.Y., NORTHRIDGE, CA, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001051184	A1	20011213
APPLICATION INFO.:	US 1999-315856	A1	19990520 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ATTN: DAVID A. FARAH. M.D., SHELDON & MAK, 225 SOUTH LAKE AVENUE, SUITE 900, PASADENA, CA, 91101
NUMBER OF CLAIMS: 115
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 4191
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The compound curcumin, derived from turmeric, inhibits phosphorylase kinase and, by doing so, exhibits a number of physiological effects related to the control of inflammation and cellular proliferation. However, curcumin is effective only when in solution. Curcumin is almost completely insoluble in water or in oils, but is soluble in alcohols. Accordingly, a method for treating inflammation in a mammal comprising administering curcumin in a solution containing at least one alcohol to a mammal to detectably inhibit the activity of phosphorylase kinase in the blood of the mammal or in a tissue of the mammal. The alcohol is preferably ethanol, 1-propanol, or 2-propanol; most preferably, it is ethanol. Instead of curcumin, a curcumin derivative or curcuminoid can be administered. The method can further comprise the administration of at least one additional compound that can be (1) vitamin D.sub.3 and vitamin D.sub.3 analogues; (2) vitamin A, vitamin A derivatives, and vitamin A analogues (3) a calmodulin inhibitor; (4) an anti-inflammatory drug; (5) a calcium channel blocker; (6) a H1 or H2 histamine blocker; (7) an antioxidant; (8) a polyphenolic compound; (9) a monoterpene; (10) genistein; (11) a soybean derived lectin; and (12) dehydrozingerone. Another aspect of the present invention is a pharmaceutical composition comprising curcumin, a curcuminoid, or a curcumin derivative in a solution containing at least one alcohol, at least one additional compound as described above, and a pharmaceutically acceptable carrier.

L5 ANSWER 8 OF 17 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001244700 EMBASE
TITLE: Prostate **cancer** chemoprevention agents exhibit selective activity against early stage prostate **cancer** cells.
AUTHOR: Liu Y.Q.; Kyle E.; Patel S.; Housseau F.; Hakim F.; Lieberman R.; Pins M.; Blagosklonny M.V.; Bergan R.C.
CORPORATE SOURCE: R.C. Bergan, Division of Hematology/Oncology, Northwestern University, Department of Medicine, 710 N. Fairbanks, Chicago, IL 60611-3008, United States
SOURCE: Prostate Cancer and Prostatic Diseases, (2001) 4/2 (81-91).
Refs: 55
ISSN: 1365-7852 CODEN: PCPDFW
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Preclinical models for the identification of prostate **cancer** chemoprevention agents are lacking. Based upon the notion that clinically useful chemoprevention agents should exhibit selective activity against early stage disease, studies were undertaken to assess whether chemoprevention agents selectively inhibited the growth of early stage prostate **cancer**, as compared to late stage **cancer**.
First, a series of cell and molecular studies were performed, which, when taken together, validated the use of a panel of prostate cell lines as a model of the different stages of carcinogenesis. Next, therapeutic responsiveness to ten different cytotoxic or chemoprevention agents was evaluated. Chemoprevention agents exhibited selective activity against normal and early transformed prostate tissue, whereas cytotoxic agents were non-specific. Selective activity against early versus advanced prostate **cancer** cells is identified as a potential screening method for chemoprevention agents.

L5 ANSWER 9 OF 17 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001293473 EMBASE
TITLE: Toxicity myths - Essential oils and their carcinogenic potential.
AUTHOR: Guba R.
CORPORATE SOURCE: R. Guba, Centre for Aromatic Medicine, 100 Dight Street, Collingwood, Vic. 3066, Australia. essentherapeutics@ozemail.com.au
SOURCE: International Journal of Aromatherapy, (2001) 11/2 (76-83).
Refs: 47
ISSN: 0962-4562 CODEN: IJARF5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
052 Toxicology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB In my previous paper, 'Toxicity Myths - the Actual Risks of Essential Oil Use' (see IJA, volume 10, issues 1&2), I considered the common 'myths' regarding the safe use of essential oils. This included discussion of often-stated 'contraindications' regarding the use of various essential oils in the case of high and low blood pressure, concerns relative to kidney and liver damage, during pregnancy and more. This paper carries on to consider further 'myths' regarding the safe use of essential oils, this time relative to the supposed carcinogenic (capable of causing **cancer**) potential of some essential oils. .COPYRGT. 2001 Harcourt Publishers Ltd.

L5 ANSWER 10 OF 17 USPATFULL
ACCESSION NUMBER: 2000:138398 USPATFULL
TITLE: Method of suppressing **tumor** growth with combinations of isoprenoids and statins
INVENTOR(S): Elson, Charles E., Madison, WI, United States
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6133312		20001017
APPLICATION INFO.:	US 1998-27546		19980223 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-39790P	19970304 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Goldberg, Jerome D.
LEGAL REPRESENTATIVE: Quarles & Brady LLP
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 1104
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of inhibiting the growth of **tumor** cells is disclosed. In one embodiment, this method comprises the step of exposing **tumor** cells to an effective amount of a composition comprising at least two compounds selected from the group consisting of tocotrienols, statins and ionones.

L5 ANSWER 11 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:521319 BIOSIS
DOCUMENT NUMBER: PREV199900521319
TITLE: Perillyl alcohol selectively induces G0/G1 arrest and apoptosis in Bcr/Abl-transformed myeloid cell lines.
AUTHOR(S): Sahin, M. B.; Perman, S. M.; Jenkins, G.; Clark, S. S. (1)
CORPORATE SOURCE: (1) Dept of Human Oncology, University of Wisconsin, 600 Highland Ave, K4-432, Madison, WI, 53792 USA
SOURCE: Leukemia (Basingstoke), (Oct., 1999) Vol. 13, No. 10, pp. 1581-1591.
ISSN: 0887-6924.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The Bcr/Abl tyrosine kinase that is expressed from the Philadelphia chromosome protects leukemia cells from apoptosis caused by removal of growth factors or by cytotoxic agents and ionizing irradiation. This resistance to apoptosis is associated with a Bcr/Abl-mediated G2/M delay. Therefore, inhibiting Bcr/Abl signaling pathways should block the ability of the Bcr/Abl kinase to protect cells from apoptosis. The monoterpenes, limonene and perillyl alcohol (POH) are new anticancer agents that selectively induce apoptosis in neoplastic cells of a variety of rodent carcinoma models. Since the potential antitumor activities of monoterpenes overlap with signaling pathways affected by the Bcr/Abl kinase, POH and limonene were tested for antileukemia activity. POH, but not limonene selectively induced G0/G1 arrest followed by apoptosis in Bcr/Abl-transformed, but not nontransformed FDC.P1 and 32D myeloid cell lines. In contrast to their greater **sensitivity** to POH, Bcr/Abl-transformed cells were more resistant than nontransformed cells to several chemotherapy agents and ionizing irradiation. Since in Bcr/Abl-transformed cells, POH induces apoptosis associated with G0/G1 arrest, POH must activate an apoptotic pathway that is not protected by the Bcr/Abl-induced G2/M delay. Monoterpenes may represent novel agents for treating Ph+ leukemias.

L5 ANSWER 12 OF 17 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999287490 EMBASE
TITLE: Isoprenoid-mediated inhibition of mevalonate synthesis: Potential application to **cancer**.
AUTHOR: Elson C.E.; Peffley D.M.; Hentosh P.; Mo H.
CORPORATE SOURCE: C.E. Elson, Department of Nutritional Sciences, University of Wisconsin-Madison, 1415 Linden Drive, Madison, WI 53706, United States. elson@nutrisci.wisc.edu
SOURCE: Proceedings of the Society for Experimental Biology and Medicine, (1999) 221/4 (294-311).
Refs: 315
ISSN: 0037-9727 CODEN: PSEBAA
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Pure and mixed isoprenoid end products of plant mevalonate metabolism trigger actions that suppress 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase activity. These actions modulate HMG CoA reductase mRNA translation and the proteolytic degradation of HMG CoA reductase. Such post- transcriptional events, we propose, are activated directly by acyclic isoprenoids and indirectly by cyclic isoprenoids. Isoprenoids, acting secondarily to the dominant transcriptional effector of sterologenesis, modestly lower cholesterol levels, if and only if, sterologenesis is not repressed by a saturating imput of dietary cholesterol. An anomaly associated with **tumor** growth - a sterol feedback-resistant HMG CoA reductase activity - ensures a pool of sterologenic pathway intermediates. Such intermediates provide lipophilic anchors essential for membrane attachment and biological activity of growth hormone receptors, nuclear lamins A and B, and oncogenic ras. **Tumor** HMG CoA reductase retains high **sensitivity** to the isoprenoid- mediated secondary regulation. Repression of mevalonate synthesis by plant- derived isoprenoids reduces ras and lamin B processing, arrests cells in G1, and initiates cellular apoptosis. This unique **tumor** cell-specific **sensitivity** allows isoprenoids to be used for **tumor** therapy, an application emulating that of the statins, but one free of adverse effects. When evaluated at levels provided by a typical diet, isoprenoids individually have no impact on cholesterol synthesis and **tumor** growth. Nonetheless, isoprenoid-mediated activities are additive, and, sometimes synergistic. Therefore, the combined actions of the estimated 23,000 isoprenoid constituents of plant materials, acting in concert with other chemopreventive phytochemicals, may explain the lowered **cancer** risk associated with a diet rich in plant products. In contrast, that lowering of **cancer** risk does not correspond to supplemental intake of other dietary factors associated with fruits, vegetables, and cereal grains, namely fiber, .beta.-carotene, vitamin C, and vitamin E, and only weakly to supplemental folate.

L5 ANSWER 13 OF 17 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999042290 MEDLINE
DOCUMENT NUMBER: 99042290 PubMed ID: 9824849
TITLE: Monoterpenes inhibit cell growth, cell cycle progression, and cyclin D1 gene expression in human breast **cancer** cell lines.
AUTHOR: Bardon S; Picard K; Martel P
CORPORATE SOURCE: Laboratoire de Nutrition et Securite Alimentaire, Institut National de la Recherche Agronomique, Jouy-en-Josas, France.. bardon@diamant.jouy.inra.fr
SOURCE: NUTRITION AND CANCER, (1998) 32 (1) 1-7.
Journal code: 7905040. ISSN: 0163-5581.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990209
AB Monoterpenes are found in the essential oils of many commonly consumed fruits and vegetables. These compounds have been shown to exert chemopreventive and chemotherapeutic activities in mammary **tumor** models and represent a new class of breast **cancer** therapeutic agents. In this study, we investigated the effects of limonene and limonene-related monoterpenes, perillyl alcohol and perillic acid, on cell growth, cell cycle progression, and expression of cyclin D1 cell cycle-regulatory gene in T-47D, MCF-7, and MDA-MB-231 breast **cancer** cell lines. Our results revealed that limonene-related monoterpenes caused a dose-dependent inhibition of cell proliferation. Of the three monoterpenes tested, perillyl alcohol was the most potent and limonene was the least potent inhibitor of cell growth. The enantiomeric composition of limonene and perillyl alcohol did not interfere with their effect on cell growth. **Sensitivity** of breast **cancer** cell lines to monoterpenes was in the following order: T-47D > MCF-7 > MDA-MB-231. Growth inhibition induced by perillyl alcohol and perillic acid was associated with a fall in the proportion of cells in the S phase and an accumulation of cells in the G1 phase of the cell cycle. Finally, we showed that the effects of limonene-related monoterpenes on cell proliferation and cell cycle progression were preceded by a decrease in cyclin D1 mRNA levels.

L5 ANSWER 14 OF 17 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97420659 MEDLINE
DOCUMENT NUMBER: 97420659 PubMed ID: 9276644
TITLE: Induction of the apoptosis-promoting protein Bak by perillyl alcohol in pancreatic ductal adenocarcinoma relative to untransformed ductal epithelial cells.
AUTHOR: Stayrook K R; McKinzie J H; Burke Y D; Burke Y A; Crowell P L
CORPORATE SOURCE: Department of Biology, Indiana University-Purdue University at Indianapolis, 46202, USA.
CONTRACT NUMBER: CA64297 (NCI)
SOURCE: CARCINOGENESIS, (1997 Aug) 18 (8) 1655-8.
Journal code: 8008055. ISSN: 0143-3334.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971024
Last Updated on STN: 19971024
Entered Medline: 19971014
AB Perillyl alcohol has antitumor activity toward pancreas and other **cancers** with low toxicity. Here, we have investigated the mechanism of action responsible for the differential **sensitivity** of malignant versus non-malignant pancreatic cells to the drug. We report that the rate of apoptosis is over 6-fold higher in perillyl alcohol-treated pancreatic adenocarcinoma cells than in untreated cells, and that the effect of perillyl alcohol on pancreatic **tumor** cells is significantly greater than its effect on non-malignant pancreatic ductal cells. Moreover, the perillyl alcohol-induced increase in apoptosis in all of the pancreatic **tumor** cells is associated with a 2- to 8-fold increase in the expression of the proapoptotic protein Bak, but Bak expression is not affected by perillyl alcohol in non-malignant cells. Thus, the antitumor activity of perillyl alcohol toward pancreatic **cancers** may be due to preferential stimulation of Bak-induced apoptosis in malignant versus normal cells. Bak may, therefore, be a useful biomarker for the chemopreventive and therapeutic effects of perillyl alcohol.

L5 ANSWER 15 OF 17 USPATFULL
ACCESSION NUMBER: 96:118614 USPATFULL
TITLE: Regression of mammalian leukemia cell **tumors**
INVENTOR(S): Gould, Michael N., Madison, WI, United States
Crowell, Pamela L., Indianapolis, IN, United States
Elson, Charles E., Madison, WI, United States
Clark, Steven S., Madison, WI, United States
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5587402		19961224
APPLICATION INFO.:	US 1995-434811		19950504 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-865561, filed on 9 Apr 1992, now patented, Pat. No. US 5414019
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Goldberg, Jerome D.
LEGAL REPRESENTATIVE: Quarles & Brady
NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Figure(s); 17 Drawing Page(s)
LINE COUNT: 580
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for causing regression of a leukemia cell **tumor** is disclosed. This method comprises the step of administering perillyl alcohol to a **tumor**-containing mammal.

L5 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 4
ACCESSION NUMBER: 1994:289616 CAPLUS
DOCUMENT NUMBER: 120:289616
TITLE: Growth inhibition of rat liver epithelial **tumor** cells by monoterpenes does not involve Ras plasma membrane association
AUTHOR(S): Ruch, Randall J.; Sigler, Kristi
CORPORATE SOURCE: Dep. Pathol., Med. Coll. Ohio, Toledo, OH, 43699, USA
SOURCE: Carcinogenesis (1994), 15(4), 787-9
CODEN: CRNGDP; ISSN: 0143-3334
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In contrast, lovastatin, a potent inhibitor of 3-hydroxy-2-methylglutaryl CoA reductase and Ras farnesylation, specifically reduced WB-ras cell growth and increased cytosolic levels of Ras. Thus, monoterpene-induced growth inhibition of rat liver epithelial cells was dissimilar to lovastatin and did not appear to involve altered Ras plasma membrane assocn. The role of altered ras oncoprotein (Ras) farnesylation and membrane assocn. in the growth inhibitory effects of several monoterpenes (limonene, perillic acid, perillyl alc., menthol, pinene and cineole) was investigated in rat liver epithelial cells. All of the above compds. except cineole inhibited the growth of viral Ha-ras-transformed rat liver epithelial cells (WB-ras cells) at concns. of 0.25-2.5 mM. These cells, however, were not necessarily more **sensitive** to these compds. compared to non-transformed and viral ras-transformed rat liver epithelial cells. Growth inhibition by limonene, perillic acid and pinene was only partially restored (20-50%) by supplementing the culture medium with 2 mM mevalonic acid. Western blot analyses of cytosolic and membranous fractions of WB-ras cells treated with monoterpenes indicated no change in Ras distribution.

L5 ANSWER 17 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1994:304924 BIOSIS
DOCUMENT NUMBER: PREV199497317924
TITLE: The chemoprevention of **cancer** by mevalonate-derived constituents of fruits and vegetables.
AUTHOR(S): Elson, Charles E. (1); Yu, Suzanne G.
CORPORATE SOURCE: (1) Dep. Nutr. Sci., Univ. Wisconsin-Madison, Madison, WI 53706-1571 USA
SOURCE: Journal of Nutrition, (1994) Vol. 124, No. 5, pp. 607-614.
ISSN: 0022-3166.
DOCUMENT TYPE: General Review
LANGUAGE: English

AB A nutritive isoprenoid constituents of fruits, vegetables, cereal grains and essential oils exhibit a spectrum of anticarcinogenic activities. The induction of hepatic Phase II detoxifying activities by dietary isoprenoids appears to underlie their blocking action. The second anticarcinogenic action of the dietary isoprenoids, suppression of the growth of chemically initiated and transplanted **tumors** is, we suggest, secondary to the inhibition of mevalonate pathway activities. Mevinolin, a competitive inhibitor of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase activity, depletes cells of the intermediate products of the pathway that are required for the posttranslational modification of proteins, a process giving the proteins lipophilic anchors that bind to membranes. As a consequence, nuclear lamins and ras oncoproteins remain in nascent states, and cells do not proliferate. gamma-Tocotrienol, perillyl alcohol, geraniol and d-limonene suppress hepatic HMG-CoA reductase activity, a ratelimiting step in cholesterol synthesis, and modestly lower serum-cholesterol levels of animals. These isoprenoids also suppress **tumor** growth. The HMG-CoA reductase of neoplastic tissues differs from that of sterologenic tissues in being markedly resistant to sterol feedback inhibition. Our review suggests that the mevalonate pathway of **tumor** tissues is uniquely **sensitive** to the inhibitory actions of the dietary isoprenoids.